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Case Report: A Case of Acute Aortic Dissection Mimicking Myocardial Ischemia

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Abstract

Acute aortic syndrome is a progressive and life-threatening disease of the aorta. Aortic dissection is caused by a circumferential or transverse tear of the intima. Initiating event may be a primary intimal tear or a medial hemorrhage that dissects into the intima. Peak incidence is in 6th to 7th decade. Presents with sudden onset of pain, the acute aortic syndrome may mimic myocardial ischemia among its variable presentations. When a patient presents with sudden onset of chest pain with electrocardiography showing ST-segment elevation, it is usually difficult to differentiate between myocardial ischemia and AAD. Here, we present a patient with chest pain with initial suspicion of myocardial infarction, AAD.

Keywords: Acute aortic syndrome, aortic dissection, myocardial ischemia, intramural hematoma.

Introduction

Acute aortic syndrome, that encompasses AAD, IM, and penetrating aortic ulcer, aortic rupture. It is defined as separation within the medial layer of the aortic wall caused by an intimal tear] (Bergmark et al., 2013). The DeBakey and the Stanford systems have been used to classify aortic dissection. The Stanford system classifies dissections that involve the ascending aorta as type A, regardless of the site of the primary intimal tear; all other dissections are classified as type B. The risk factors associated with AAD include hypertension, atherosclerosis, known aneurysm, Marfan syndrome, Loeys deitz syndrome, ehler danlos syndrome (Corvera, 201).

Dissection of the ascending aorta is more common than descending aorta. Intramural hematoma is a hematoma within the medial layer of the aortic wall without intimal injury. Patients with IM are usually of 5th to 6th decade, it is more commonly present with aortic aneurysm, usually occurring in patients with severe atherosclerotic disease and rarely in those with Marfan syndrome. Intramural hematoma is generally held to account for between 5–20% of patients admitted to the hospital diagnosed with AAS or AAD (Xie et al., 2014).

Quick and accurate diagnosis of acute aortic syndromes are difficult due to the wide variety of clinical presentations such as acute coronary syndrome, gastrointestinal disease (such as cholecystitis or pancreatitis), musculoskeletal disease and respiratory diseases (such as pulmonary embolism). CT scan and MRI scan remain the gold standard to diagnose intramural hematoma. Computed tomography scan with intravenous contrast is vastly available and can be performed rapidly in most emergency departments, it has a sensitivity of 95% and specificity between 85–100%. Magnetic resonance imaging scan has a sensitivity and specificity of 100% (Longe, 2008).

Case Report

A male pt' of age 65yrs came with sudden onset of severe chest pain.

Pain is of radiating type, radiating to the left shoulder, shortness of breath is present.

ECG was taken. It showed ST elevations, biphasic T waves in V1 to V4 leads.

VITALS are like bp is 90/60 mm of hg, pr is 62 bpm afebrile.

Cardiac catheterization revealed no infarcts and it is normal.

Trans thoracic Echocardiography revealed aortic dissection.

CT scan was done. It revealed an acute Intramural hematoma and aortic dissection (Stanford type B).

Course

The patient was treated with IV labetalol infusion for blood pressure control (less than 120).

A review CT scan was performed after 24 hours, showing decrease in intramural hematoma.

The patient's IV blood pressure medications are changed to oral antihypertensive medications with labetalol, losartan.

Outcome

After the pt' is free of symptoms he was discharged with close follow-up.

Uncomplicated IM confined to the descending aorta are treated medically with ICU monitoring, blood pressure and pain management.

Results and Discussion

Acute aortic syndrome, that encompasses AAD, IM, and penetrating aortic ulcer, aortic rupture. It is defined as separation within the medial layer of the aortic wall caused by an intimal tear]. The DeBakey and the Stanford systems have been used to classify aortic dissection. The Stanford system classifies dissections that involve the ascending aorta as type A, regardless of the site of the primary intimal tear; all other dissections are classified as type B. The risk factors associated with AAD include hypertension, atherosclerosis, known aneurysm, Marfan syndrome,Loeys deitz syndrome,ehler danlos syndrome. . Dissection of the ascending aorta more common than descending aorta. Intramural hematoma is a hematoma within the medial layer of the aortic wall without intimal injury. Patients with IM are usually of 5th to 6th decade, it is more commonly present with aortic aneurysm, usually occurring in patients with severe atherosclerotic disease and rarely in those with Marfan syndrome. Intramural hematoma is generally held to account for between 5–20% of patients admitted to hospital diagnosed with AAS or AAD.

Quick and accurate diagnosis of acute aortic syndromes are difficult due to the wide variety of clinical presentations such as acute coronary syndrome, gastrointestinal disease (such as cholecystitis or pancreatitis), musculoskeletal disease and respiratory diseases (such as pulmonary embolism). CT scan and MRI scan remain the gold standard to diagnose intramural hematoma. Computed tomography scan with intravenous contrast is vastly available and can be performed rapidly in the most emergency department, it has a sensitivity of 95% and specificity

between 85–100%. Magnetic resonance imaging scan has sensitivity and specificity of 100% Intramural hematoma is best seen on non-enhanced computed tomography scan. It appears as an area of hyperattenuating crescentic thickening in the aortic wall, which was seen on the CT scan findings of our patient.

Once diagnosed, treatment options depend on disease severity. Acute dissections involving the ascending aorta are considered surgical emergencies. In contrast, IM confined to the descending aorta are treated medically unless there is a high risk of end-organ ischemia or continued hemorrhage into the pleural or retroperitoneal space, which can be treated with an endovascular approach. Acute uncomplicated type B dissection should be treated with medical therapy including intensive care unit monitoring, reasonable control of systolic blood pressure to 100–120 mmHg and pain control. Beta-adrenergic antagonists (esmolol, metoprolol, or labetalol) represent the first-choice agents, and vasodilators (sodium nitroprusside) and calcium channel antagonists can be used.

In our case, the patient was treated appropriately with aggressive blood-pressure control with intravenous beta-blocker labetalol infusion as it is a drug with both alpha and beta-adrenergic blocking properties. Long-term therapy includes long-term control of HTN, with a beta-blocker plus a ccb or ACEI and should be followed on OP basis for every 6 -12 months.

Conclusion

High clinical suspicion and timely diagnosis are crucial to prevent the devastating sequelae of the acute aortic syndrome. Therefore, it is crucial to have a high index of suspicion for acute aortic dissection in cases of chest pain. In addition, clinicians should consider that aortic dissection is one of the differential diagnoses for acute chest pain syndrome, even in the presence of typical electrocardiogram changes for acute myocardial infarction.

Author contribution

Vishnuvarthan R S, Valarmathy M, Suresh Babu P, Karthikeyan C T encouraged and supervised the findings of this work. All authors discussed the results and contributed to the final manuscript

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Conflict of interest: Nil

Study significance

Patient presents with sudden onset of chest pain with electrocardiography showing ST segment elevation, it is usually difficult to differentiate between myocardial ischemia and AAD. Here, we present a patient with chest pain with initial suspicion of a myocardial infarction, which was AAD.

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